for 70 CAD is improved: 3) This now easily discernible qualitative marker improves the ability of the perfusion study to reliably identify 70 CAD.

1103-147 Are the SPECT Myocardial Perfusion Patterns Different in Ischemic vs Non-Ischemic Cardiomyopathies?

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To characterize and compare the SPECT scintigraphic patterns in ischemic (I) and non-ischemic (NI) cardiomyopathies (C), the stress T1-201 SPECT images of 37 patients with known C (24 I, 13 NI) were reviewed. Tomograms were analyzed using the 20 segment scoring system for Stress extent score (SES = number of abnormal stress segments), severity score (SEV = % maximal counts in worst defect), reversibility score (REV = stress minus delayed ES), ejection fraction (EF), SES / EF ratio (SES/EF), lung / heart ratio (L/HR) and presence of LV dilation at stress (LVD).

Results: The univariate analysis of the differences between IC and NIC is displayed on the table. By multivariate analysis, the best predictor of IC Vs NIC was a high SES ($p = 0.0001$) or a high SES/EF ($p = 0.0002$) followed by an increased L/HR ($p = 0.04$). EF was not a significant predictor. By ROC analysis of all variables, the best criterion to discriminate IC from NIC was a SES ≥6 or an SES/EF ratio ≥0.20, with 88 and 85% correct classifications into IC and NIC, respectively (e.g. a SES of 6 with an EF of 20% results in a SES/EF ratio of 0.3). *Thus*, SPECT patterns differ in IC compared to NIC: for a similarly depressed LVEF, perfusion defects are more extensive and more severe with ischemic Vs non-ischemic cardiomyopathies. Lung uptake is greater with the former. These criteria offer promise for identification of ischemic Vs non-ischemic cardiomyopathies in a prospective population.

	NIC	IC	p =		NIC	IC	p =
% EF	37 ± 7	31 ± 8	0.03	REV	6.7 ± 5	8.3 ± 6	ns
SES	3.7 ± 3	8.6 ± 3	0.00001	L/HR	42 ± 9	64 ± 20	0.0001
SEV	55 ± 9	34 ± 16	0.0001	LVD	0/13	8/24	0.03
SES/EF	0.1 ± 0.08	0.3 ± 15	0.0001				

1103-148 Memory Function of Fatty Acid Metabolism Imaging for Detecting Post-ischemic Myocardium in Unstable Angina: A Comparison With ECG Changes and Wall Motion

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We have previously shown that I-123 beta-methyl iodophenyl pentadecanoic acid SPECT (BMIPP) is useful in locating the culprit-lesion vessel territories in unstable angina (UA). To examine how long ischemic cardiac "memory" detected by BMIPP persists, we compared rest BMIPP with ECG changes and wall motions in 55 patients (pts) (mean 62 years, male/female = 44/11) with no previous MI who admitted within 24 hours after episodes. After stabilization of symptoms by medical therapy, BMIPP and coronary angiography were performed mean 7.0 ± 4.8 days and 6.8 ± 4.3 days after onset. ECGs were obtained at least once daily during the first 7 days and at the time of BMIPP and coronary angiography /left ventriculography. Thirty-eight pts had sudden or new onset angina and 17 worsening pattern of angina. All pts had significant coronary disease (n = 40) or induced vasospasm (n = 15) documented by angiography. Culprit lesions or vasospastic arteries were the left anterior descending artery in 35 pts, right coronary artery in 15, circumflex artery in 3 and undetermined in 2. Abnormal ECGs (i.e.T inversion, ST segment elevation or depression) were found in 34 (62%) pts at admission, and normalized in 12 and newly developed in 6 through the clinical course. Finally 28 (51%) pts had abnormal ECGs at the time of BMIPP. Detection of post-ischemic myocardium was 39/55 (71%) by BMIPP ($p < 0.05$ vs. ECG and $p < 0.0001$ vs. wall motions), 28/55 (51%) by ECG and 12/55 (22%) by regional wall motions mean 7 days after onset.

Conclusions: Depressed fatty acid uptake persists a week and longer than ST-T changes and is found in pts without ECG changes. Thus, BMIPP as ischemic cardiac memory imaging is a clinically useful method to detect post-ischemic myocardium in pts with UA even after stabilization of symptoms.

1103-166 Severe Coronary Artery Disease in Patients With Normal or Near Normal Myocardial Perfusion Stress Imaging

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It is accepted that normal or near normal myocardial perfusion stress imaging with T1-201 or Tc99m-Sestamibi suggests the absence of life threatening coronary artery disease (CAD). We reviewed myocardial perfusion studies from the past 5 yrs. in order to (1) define the proportion of false negative results, and (2) ascertain the features which may prevent misdiagnosis. Out of 9171 tests, 3992 (44%) were interpreted as normal or near normal; 97 (1%) patients subsequently (within 6 months) underwent coronary angiography. Seventy (22M/48F; 58 ± 13 yrs.) had no significant CAD. Eight (5M/3F; 65 ± 8 yrs.) had either left main (N = 3), severe proximal LAD (N = 3) or 3 vessel CAD (N = 2).

	No CAD N = 70	Severe CAD N = 8
Exercise/pharmacologic (pts.)	43/27	5/3
T1-201/Tc99m-Sestamibi (pts.)	59/11	4/4
Artifacts (soft) tissue attenuation, "hot spots") (pts.)	24	5
Markers of severe CAD* (pts.)	25	7**

* lung uptake and/or transient cavity dilatation with T1-201, ST-segment depression, chest pain and or hypotension with exercise, ** $P < 0.01$ No CAD vs. Severe CAD, 2-tail Fisher's Exact Test, PPV = 7/32 (22%)

The peak heart rate achieved (exercise studies) was 89% and 87%, respectively. Although both groups had similar proportion of artifacts, patients with severe CAD had significantly more markers of severe CAD ($P < 0.01$). Thus, the referring physician should be aware of the possibility of significant CAD in patients having normal or near normal perfusion stress imaging accompanied by known markers of severe CAD.

1104 Hypertension and Endothelial Function

Tuesday, March 31, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.–11:00 a.m.

1104-47 Systemic Nitric Oxide Production in Insulin-resistant and Insulin-sensitive Male Subjects With Essential Hypertension

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Insulin resistance (IR) often coincides with endothelial dysfunction. In addition, insulin-dependent glucose transport and insulin-mediated NO production share common elements in signal transduction. Our aim was to assess if systemic NO formation and its response to acute hyperinsulinemia are related to IR in essential hypertension (EH).

Whole body insulin sensitivity (M) was measured using the hyperinsulinemic euglycemic clamp in 17 untreated non-obese male pts (age: 39 ± 10 years) with newly diagnosed, uncomplicated, pure EH and 10 healthy controls. Urinary excretion rates of NO_2^- plus NO_3^- (Uno_x) and cyclic GMP (U_{cGMP}) (corrected by urinary creatinine level) were compared in basal conditions and during the clamp in 10 insulin-resistant EH pts (M < 5.9 mg/kg/min) (group A), 7 insulin-sensitive EH pts (group B), 10 insulin-sensitive controls (group C).

The 3 groups did not differ in age, body mass index, blood glucose and cholesterol. Uno_x was lower in groups A and B vs C ($p < 0.05$) (A: 53 ± 17; B: 61 ± 18; C: 81 ± 19 μmol/mmol creatinine). Relative Uno_x changes on the clamp (ΔUno_x) exhibited high interindividual variability and no intergroup differences (A: 17 ± 39; B: 8 ± 27; C: 3 ± 22%). Uno_x and ΔUno_x did not correlate with M either in any group or pooling all data together. Uno_x and U_{cGMP} were correlated ($r = 0.62$, $p < 0.01$ for all data) and their time course was similar.

Conclusion: In EH systemic NO formation is depressed irrespective of insulin sensitivity. The response of NO production to acute hyperinsulinemia is heterogenous and unrelated to insulin sensitivity.

1104-48 Endothelial Vasomotor Dysfunction in Offsprings of Patients With Essential Hypertension

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Background: It is documented that endothelium dependent vasodilation is impaired in patients with essential hypertension. The aim of this study was to identify any early vascular changes that may be predictive of future hypertension in young subjects with family history of essential hypertension.

Methods: The study was conducted on 23 normotensive young males (26.6 ± 14.6 years old). Subjects were divided into two groups; 12 without family history of essential hypertension (FH (−)) and 11 with family history (FH